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Epidemic Evolution: Source, Lock-down and Removal Modifications to the SIR Model

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Key words: modified SIR model, epidemic, death,lockdown source.

Abstract

The original purpose of this article was to modify the original SIR equations to allow for a direct source of infection (without which the original equations would have no solutions unless one starts with an already infected population) and also to see to what extent one could obtain multiple outbreaks of an infectious disease. In the course of developing the basic ideas several other factors arose to take prominent roles.

Perhaps one of the more salient factors is the point that choosing an arbitrary time to change conditions from say a lock-down for the population to a less stringent social behavior, such as allowing partial or complete opening of businesses and schools, etc. should be based on knowledge of the disease and its evolution. Such decisions are usually made by politicians who have less than full information concerning the consequences of their actions. Several examples are given to illustrate these points.

Introduction

In the almost last 100 years a concerted effort has been undertaken to describe quantitatively the temporal evolution of epidemic and pandemic diseases (Kermack and McKendrick, 1927) perhaps triggered by the devastating pandemic of the so-called Great Influenza which ravaged the world for over three year starting around 1918 and for which estimates put the world death toll at around 50-100 million people (Barry, 2004), although there is considerable uncertainty on even these estimates. Since then there have been numerous quantitative models suggested to describe the evolution of infectious diseases. A good summary is provided by Hethcode (2000).

Despite the progress made with such models the main difficulty is not so much in the mathematical equations *per se* but in the use of "blocks" to describe the epidemiology without due concern being given to the underlying causes of the block parameters. Perhaps such a

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description is inevitable but it leaves one with less than complete satisfaction with such models.

Here we consider again the basic model of Kermack and McKendrick (1927) to investigate solution behaviors The major point to be addressed is to see to what extent solutions are influenced by both the combined choices of parameters in the blocks and the initial conditions. The non-linearity of the basic equations is the main cause of the interaction of parameter choices and initial conditions, leading to constraints that would not otherwise obtain. Such an investigation points the way to a more complete description of model behaviors and so indicates the potential for obtaining quantitative results more closely in accord with observations.

There are just three groups of factors in the basic model of Kermack and McKendrick, (1927): Susceptible (S) people, Infected (I) people and Removed (R) people. Thus if infected then eventually one must recover – something that seems anathema to all know cases of epidemic patterns. Thus there is no allowance made for deaths from a pandemic (or epidemic) disease nor is there allowance made for a fraction of the "Removed" population to be thereafter immune, nor for the possibility that the remaining fraction of the "removed" population could again be infected. Further the basic equations describing the evolution do not contain a source of infection so that should there be no infected people at the start of the pandemic then there are never any – such a behaviors requires one to insert an extra mechanism somehow into the epidemic equations to describe, at the very least, the start of infection

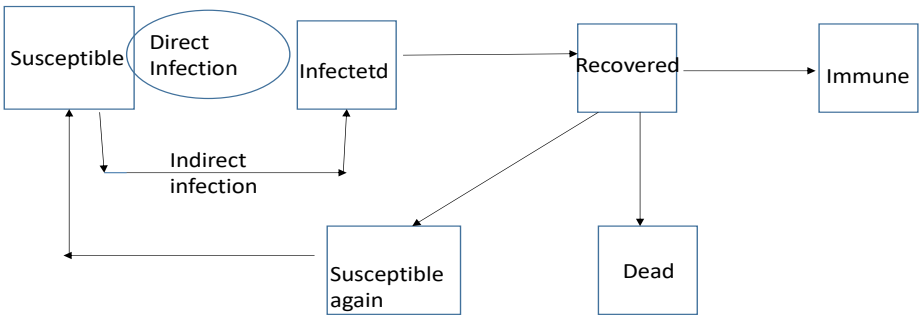

Figure 1. Flow diagram indicating the evolution of disease.

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The basic SIR equations are given in the Appendix so as not to break the thread of the arguments to be presented. In addition the modifications to the basic equations are also given so that one can follow their impact on functional behavior. The point here is to show that seemingly simple changes to the SIR equations produce major changes in the temporal behavior of solutions.

There is also concern to see to what extent changing conditions during an epidemic or pandemic has on the spread and intensity of disease. For instance forcing a population "lock-down" is one way of slowing the spread of a disease but the lock-down must be complete for otherwise a fraction of the population is still free to move around and can either catch the disease or, if infected already, pass the disease on to others. Thus the so-called "partial lock-down" leaves massive loop-holes for a disease to continue to spread. It is all very well for individuals (who are then healthy) to complain about being in a lock-down situation but the alternative is that they do not follow lock-down orders or suggestions and so end up sick or, even worse, dead and take others with them down the same path.

The sense of the present article is to show two major aspects.

First to discuss the structural pattern of disease evolution for only a single wave of a disease, i.e. in the model behavior all parameters (a, b, μ) are held constant and the initial conditions (at t=0) are that S=1,I=0.

Second to discuss the structural pattern of disease evolution when the parameter a is changed to a value A at normalized time T* and thereafter held constant while the parameters a, b, and μ are held constant from time t = 0 through to time t*=T*/a with the initial values again S=1, I=0 at t=0,and that I and S be continuous at time t* = T*/a. As will be seen directly such changes allow one to produce results similar to observed disease changes when lock-down conditions are changed. The mathematical development is given in the Appendix so as not to break the flow of discussion here in the main text. Only relevant details are presented here and readers are encouraged to check the Appendix for fine details. Consider each aspect in turn.

A. Structural Pattern of Disease Evolution for a Single Epidemic Wave

The sense here is to present the temporal structure of the disease evolution eschewing any considerations of variable parameters, mutations or other extraneous factors. The point is to provide a basic structure as a template on which one can impress the effects of other changes as will be discussed in subsection B below.

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Perhaps the important point to make is that the pattern of behavior is given through I=bJ/a and S =bx/a to obtain the form

$$dJ/dx = -1 + qJ/(x*(J+1)) \quad (1)$$

Here one also has time, t, given through dx/dt = -bx(1+J) with normalized time T as T =bt as also derived in the Appendix.

The critical point to note is that the structure of J depends on only the single parameter q (the ratio of the frequency of loss from the infected fraction to the frequency with which the susceptible fraction are directly infected .i.e. q = μ/b) the so that the factor b/a connecting I and S to J merely change the magnitude scale but not the structure. It is therefore more than adequate to describe the evolution in terms of J(T) with later scaling to I and S mutatis mutandis if deemed necessary.

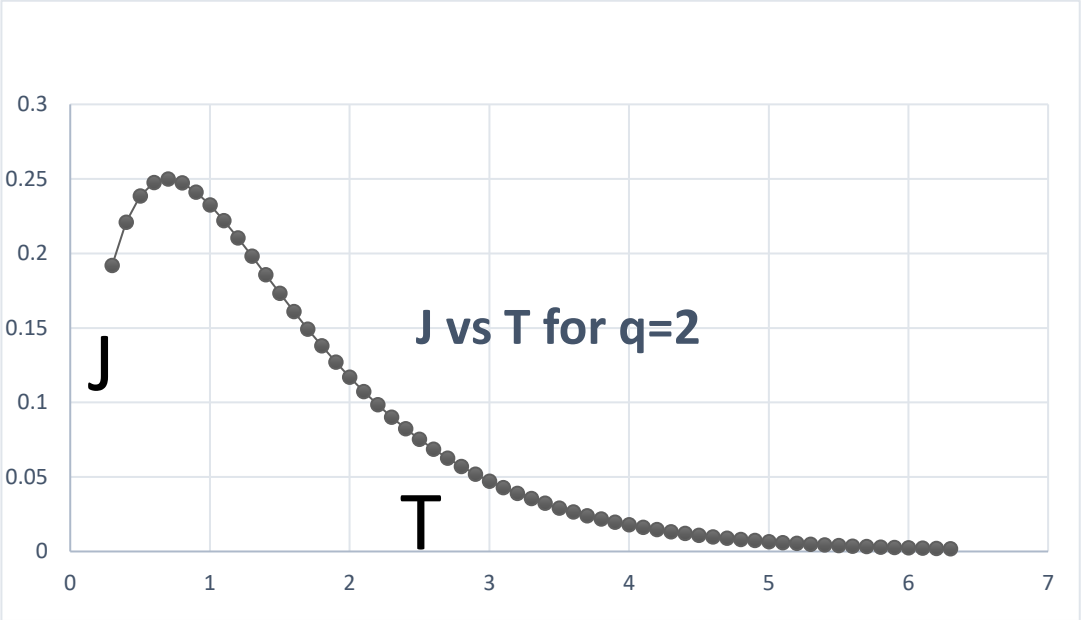

Figure 2. Representative case of the evolution of J with normalized time T for q=2.

Figure 2 shows a representative case of the evolution of J with normalized time T for q=2 while figure 3 shows the evolution for a smaller value of q =0.9. One notes that for smaller q values the peak of infected situations is larger than for q= 2 and the time to have the decay of the disease is increased. This variation is to be expected because q=2 represents a larger removal rate compared to the situation with q=0.9.

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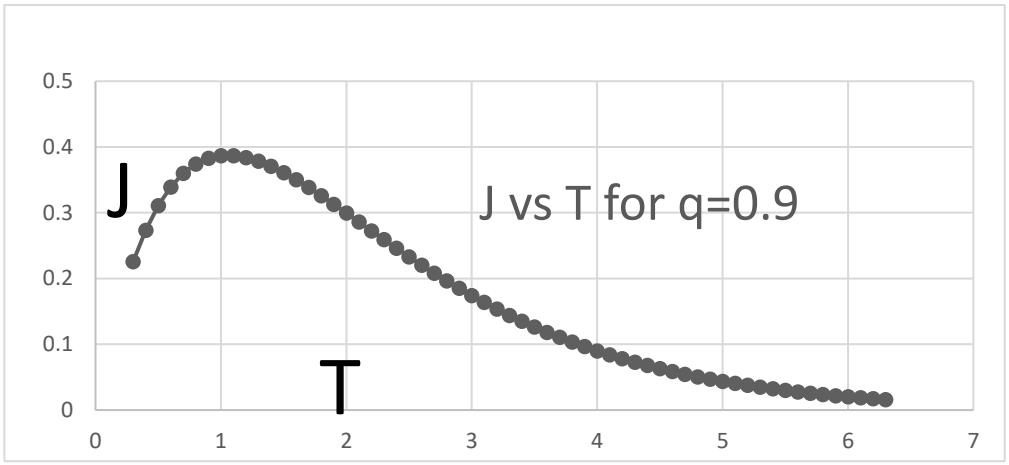

Figure 3. Representative case of the evolution of J with normalized time T for q=0.9.

One can also note that the peak value for J (J_{max}) as well as the normalized time (T_{max}) at which the peak is reached both decrease as q increases (as shown in figures 4 and 5 respectively), again because the rate of loss of infected people is larger as q increases.

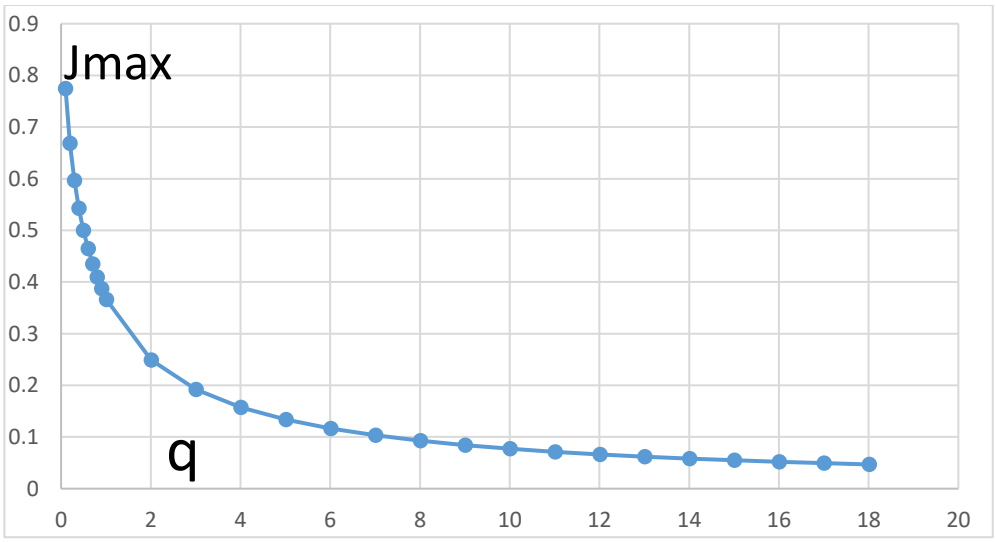

Figure 4. The peak value for J (J_{max}) decreases as q increases.

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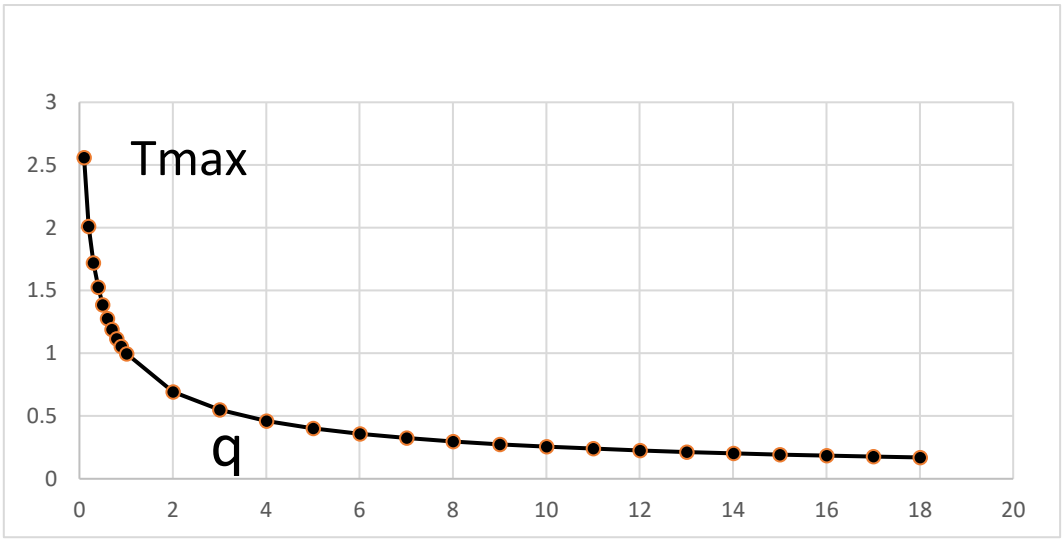

Figure 5. The normalized time (T_{max}) at which the peak for J is reached decreases as q increases.

B. Structural Pattern of Disease Evolution for Parameter Changes with Time.

Guided by the behavior of J(T) for a single wave of epidemic evolution consideration can now be given to situations where there are changes in the conditions pertaining to the disease evolution. Three situations exemplify the general patterns of possible behavior.

Shown in Figure 6 is the change in J with normalized time when q =10, corresponding to rapid removal of infected people but with a change in a to 1% of the value that pertained before normalized time T =5.5. One sees that the choice of timing for the change is dominantly responsible for the long slow decline after time T= 5.5 due to the small value of 1% now chosen for a shift in a at time 5.5.Note that while the second wave is not quite as high as the first wave it is longer lasting so more people are infected in total.

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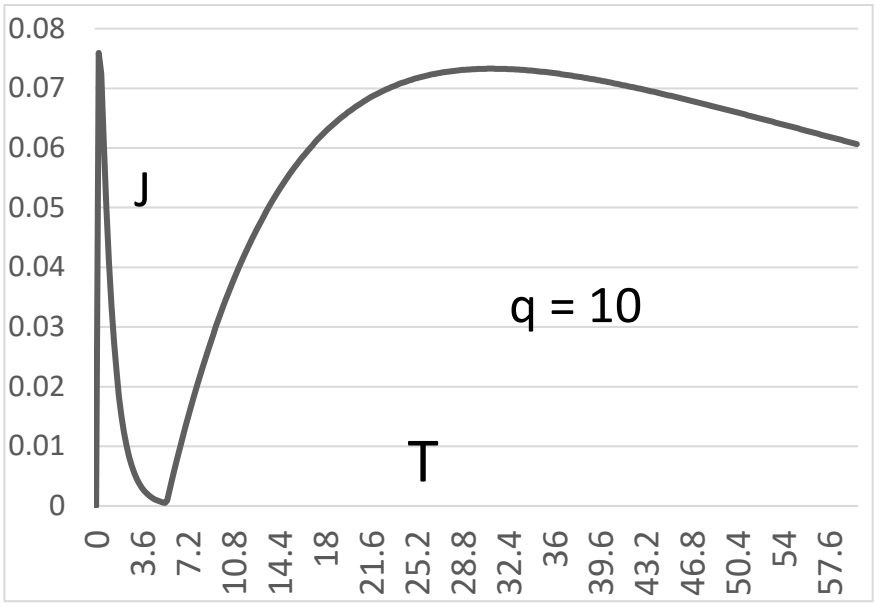

Figure 6. Change in J with normalized time when q =10, corresponding to rapid removal of infected people but with a change in a to 1% of the value that pertained before normalized time T =5.5.

Now arrange that one lowers q from the value 10 shown in Figure 6 to a value 1.8, representing a slower removal of people from the infected domain, and also let the value a be 0.2 of its value that pertains to normalized time T<7.2. In this way one delays a change in parameters to later than in Figure 6. Then note from Figure 7 that one has two sharp peaks both of which are larger than the corresponding peaks in Figure 6 and so represent more infected people because the removal is much lower (q = 1.8 compared to q=10) and that the second wave is more rapidly decreased than the corresponding second wave in Figure 6.

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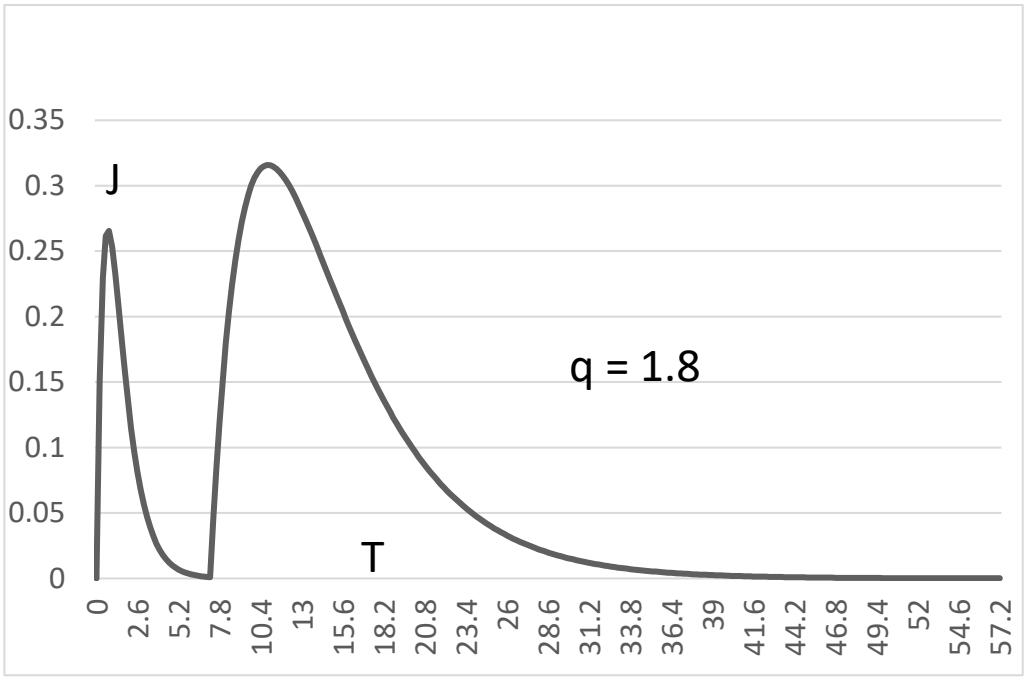

Figure 7. Lower q from the value 10 shown in Figure 6 to a value 1.8, representing a slower removal of people from the infected domain, and also let the value of a be 0.2 of its value that pertains to normalized time T<7.2. In this way one delays a change in parameters to later than in Figure 6. Then note that one has two sharp peaks both of which are larger than the corresponding peaks in Figure 6 and so represent more infected people because the removal is much lower (q = 1.8 compared to q=10) and that the second wave is more rapidly decreased than the corresponding second wave in Figure 6.

Figure 8 shows the situation where the time for change in parameter values is reduced to T = 4.5, and the removal rate is increased to q= 5 with the value a being set at 10% of the worth for times <4.5. Then one notes that while there is a second peak it is significantly less than the first peak indicating that there has been some success in controlling the disease although there is still the long slow “tail” of decline.

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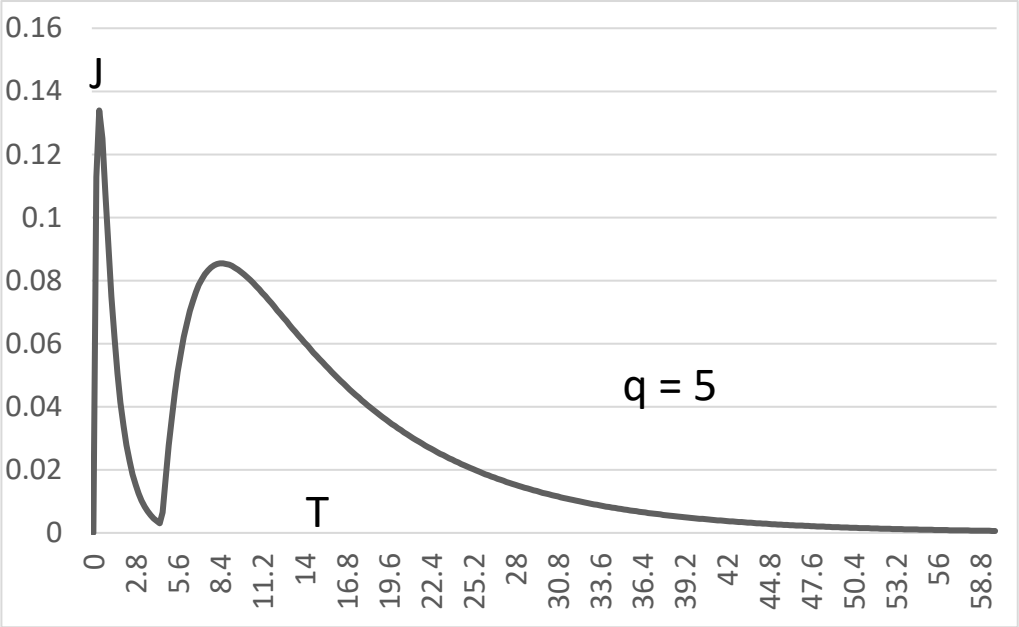

Figure 8. The situation where the normalized time for change in parameter values is reduced to T = 4.5, and the removal rate is increased to q = 5 with the value a being set at 10% of the worth for times <4.5. Note that while there is a second peak it is significantly less than the first peak indicating that there has been some success in controlling the disease although there is still the long slow "tail" of decline.

These three simple situations show that a judicious choice of timing and values for parameter changes can have either a deleterious effect on the fraction of the population that sickens (or dies) but, equally, can have a beneficial effect when applied with knowledge and concern of the virulence of the disease be it viral or bacterial.

While only a single time for change in parameter values has been described here it is clear that one could repeat the process and so obtain an estimate of which parameters are most important to know with precision if one were to be concerned with multiple changes- such as repeated lock-down and easing of conditions to try to ameliorate disease spreading.

C. Removal, Recovery. Death, Immunity, Mutation and Further Susceptibility.

While the basic patterns of behavior described here give one a basis to assess the ebb and flow of an infectious disease they are far from a complete picture. The point is that many factors influencing the viral response have been totally neglected or, at best, given only a cursory glance. For instance removal of people from the infectious group is described through $dR/dt = \mu(t)I$. The removal can be split into different categories: Removal due to death from the disease; removal due to immunity; removal due to ultimate recovery from the disease, or

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removal due to recovery but with the chance of being, again, part of the susceptible fraction so that one can be infected once more. None of these individual factors has been considered here because to do so means that one needs more parameters to describe the various categories. Such information is likely available only from studies involving actual case histories.

Equally the question of mutation of an infectious disease is always a concern. It is all very well to make significant progress at understanding the spread of an original disease but if the disease mutates at some later time (say to a more virulent form that is easier to infect) then the question is always to what extent has the work done at understanding the evolution of the original disease been of worth. This aspect of disease propagation is also not considered here but is of considerable concern for when a disease mutates, how often, and whether some of the mutated forms are deadlier than the original are matters of significance. However without more information to assess the parameters needed that are involved in mutation the task is less than simple.

Discussion and Conclusion

While the original purpose of this article was to modify the original SIR equations to allow for a direct source of infection (without which the original equations would have no solutions unless one starts with an already infected population) and also to see to what extent one could obtain multiple outbreaks of an infectious disease, in the course of developing the basic ideas several other factors arose to take prominent roles.

Perhaps one of the more salient factors is the point that choosing an arbitrary time to change conditions from say a lock-down for the population to a less stringent social behavior, such as allowing partial or complete opening of businesses and schools, etc. Such decisions are usually made by politicians who have less than full information concerning the consequences of their actions. Indeed, it happens that some such decision is taken and then somewhat later one notes that the infected population has risen rapidly so one then invokes a second stringent lock-down in order to limit the spread of the disease. Perhaps such actions are inevitable but surely could be ameliorated with some foresight. The consequences of such "second waves" are increased deaths.

What is also clear is that mutation is a very common event for any disease and this fact alone means that attempts to control the spread of a disease are hampered unless one can devise a multipurpose vaccine against all the mutation forms- something mankind has not yet

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been able to do with either the common cold or influenza so that more recent infectious diseases that mutate would seem not likely to be brought under control either.

Perhaps the most glaring omission from the quantitative development is the fact that there is nowhere any consideration given to spatial evolution of a disease. For instance the great influenza of 1918 started somewhere in Kansas and spread throughout the world because of troop transport (Barry, 2004) or the Civid-19 virus that seems to have started in Wuhan, China and from there spread through many channels throughout the world (WHO, 2021). The point is one needs a spatial component to the equations describing spread of an infectious disease be it from bacilli or viral in origin. Yet nowhere, even in the original SIR equations, is there any consideration given to spatial and temporal spread. Perhaps such an omission is necessary to understand even a fraction of infectious disease evolution but it would seem difficult to comprehend the spread of a disease without inclusion of the spatial character. Accordingly while the present article seems to be including factors relevant to modeling how an infectious disease develops with time one still has a very long way to go in order to have a more complete quantitative understanding of such matters.

Finally note that while only one or two waves of disease evolution have been described one could just again change the conditions for lockdown versus non-lockdown say and so promulgate as many waves as one deems necessary. Lastly while the development given here has been done for constant parameter values there is no fundamental difficulty in considering variable parameters. The requirement is that one knows how to vary the parameters and for that one needs a more detailed epidemiology than considered with “bloc” modeled behaviors as given here for there is no obvious reason that an infectious disease should feel honor-bound to behave as a model would have it.

There is indeed a long road to hoe before we have a decent understanding of the spread of infectious diseases.

Appendix

a. The original SIR equations

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The original equations show that there is no solution unless one starts with an infected population:

$$dI/dt = a(t)SI - \mu(t)I \quad (A1)$$

$$dS/dt = -a(t)\,SI \quad (A2)$$

The infected (I) fraction of the population, the susceptible (S) fraction are the basic variables and the parameters a(t) and μ(t) describe the epidemiological behavior in block form. The removed population fraction (R) is then given through

$$R = 1-S-I \quad (A3)$$

One sees immediately that if I = 0 at time t = 0 then I is forever after zero so there is no infection. Accordingly, somehow a "start" value for I must be included. One also notes that even if there are values for S and I then the only logical end product is that all infected people eventually recover, which is not what real epidemics and pandemics do- real people die! So one has to modify the basic SIR equations and that is the task of the next section of this Appendix.

b. Modifications to the SIR equations

Modifications have to be made in consideration of real epidemic behavior. To do so most simply is to appeal to a couple of known cases that exemplify the patterns of epidemic behavior.

Consider briefly the London plague years (1664-1667) of the Middle Ages. It is known (Defoe, 1772) that the direct cause of plague was the fleas carried by rats that, once their rat hosts started to die, then jumped to the next available source of blood, viz. humans. The injection by the fleas of the botulism toxin then rapidly caused human plague-infected people who, in turn, could then pass the plague to others. In this case the start of plague in humans was attributed to contact with flea-carrying rats. People died from the toxin either in pneumonic or bubonic form. Some fraction of the population survived of course.

Consider, also briefly, the great influenza pandemic of 1918-1923. It is thought (Barry, 2004) that the start of the pandemic was due to human contact with farm animals that had been infected by an influenza virus of surprising virility against humans. (there are many such influenza viruses so that mutations are common even today). The spread of the influenza was then carried out rapidly through human-to-human contact notably at first at the many induction forts for volunteers for the First World War but then by the transfer of such troops to European theaters of operation and to the civil populations. The best estimates, while rough, suggest that between about 50-100 million people died world-wide from that influenza episode with around half dying in the first year (Barry, 2004). So again one has a situation where the primary source of human epidemic is the transfer to humans from some

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other species with later direct human-to-human contact. At the present time vaccines against influenza are available in the forms of neuraminidase inhibitors but are, nevertheless, not 100% effective.

Around the world one has a yearly outbreak of influenza (of Types A, B, or C –Type D does not attack humans seemingly) with about 3-5 million people infected and between around 300-600 thousand deaths.

What one needs to do is to incorporate these two effects at least into the SIR equations. Simple methods of doing so are available. One can note that by adding a source factors b(t)S to the right hand side of equation (A1) one covers the contingency that the infected rate at the outbreak time (where S=1 and I = 0) is just b(0) and so one has what one needs to make the equations describe correctly an outbreak. As with the other bloc parameters in the SIR equations the factor b(t) describes the transfer of the epidemic to humans from elsewhere

One also needs to incorporate the death rate of people because otherwise the only end situations of the SIR equations, including the modification bS, is that the infection rate tends to zero and all infected people have recovered in contrast to observed epidemic and pandemic situations. A simple procedure to do so is to take the removed rate, R, and replace it by fR, where f is a fraction and may be time dependent. Then fR (≡ D) is the fraction of the population that died at time t. Indeed one can then take a second fraction g of the removed population as being immune and so of no further interest from the point of view of spread of the disease- the exception being a “Typhoid Mary” situation (See Note in references) where a person carries the disease and is able to infect others while remaining completely fit oneself. The remaining removed fraction, (1-f-g)R, is then composed of those who have truly recovered and can function again. Figure 1 shows a corresponding flow diagram for an epidemic or pandemic.

In short one writes the modified equations in the form

$$dI/dt = a(t)SI + b(t)S - \mu(t)I \qquad (A4)$$

$$dS/dt = -a(t)\,SI - b(t)S \qquad (A5)$$

and by adding equations (A4) and (A5) one has

$$d(D+fR)/dt = \mu(t)I \qquad (A6)$$

with $$S+I+fR+D = 1 \qquad (A7)$$

One should be aware, of course, that missing in these modifications are many effects such as the influence of diet, gender, age and so on. However the most serious defects of the original SIR equations are corrected and it is then of interest to see what impact the modifications make to solutions to the equations. That mathematical development is the next part of this Appendix.

c. Mathematical Manipulations with the modified SIR equations

Divide equation (A4) by equation (A5) to get

$$dI/dS = -1 + \mu(t)I/(S*(b+aI)) \qquad (A8)$$

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If μ, b and a are all constant then substitute I =bJ/a and S =bx/a to obtain the form

$$dJ/dx = -1 + qJ/(x*(J+1)) \quad \text{(A9)}$$

where q = μ/b. One also has

$$dx/dt = -bx(1+J) \quad \text{(A10)}$$

Now one knows that J = 0 on S=1, the start of the pandemic, so that at least in the small time domain around t =0 one can ignore the factor J in the expression J+1 of equations (A9) and (A10). Under such a neglect the general solution to the pair of equations is simply

$$J = (\exp(-T) - \exp(-qT))/(q-1) \quad \text{(A11a)}$$

$$x = (a/b) \exp(-T) \text{ (so that S=1 on T=0)} \quad \text{(A11b)}$$

with T =bt. (The exceptional value q=1 in equation (A11a) yields J = Texp(-T)).

The maximum value of J occurs on T* = -lnq/(1-q) with

J (max) = (exp(-T*) –exp(-qT*))/(q-1). Plots of these behaviors are given in figures 4 and 5.

Note in particular that the structural shaping of J versus x is dependent on only the one parameter q plus of course the boundary conditions at time t =0.

d. Change in parameters at a specified time

In order to account for more than one outbreak of a viral pandemic it is relatively simple matter to argue as follows. Suppose that at a specific normalized time (T = T* with real time t* =T*/a) one were to change the lock-down conditions either to more stringent or to more lax. Then the parameter a in equation (A4) would be changed immediately. This simple change allows one to model a multi-outbreak. For if a is changed to a value A at normalized time T* and thereafter held at that value then there is a different chance of infections. After time normalized time T* then the basic equations are again in play but with values at time t=T*/a for both I and S rather than the values of S=1 and I = 0 pertaining at time t= 0. A discussion of this effect is given in the main text too.

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Typhoid Mary, real name Mary Mallon, (born September 23, 1869, Cookstown, Ireland—died November 11, 1938, North Brother Island, Bronx, New York, U.S.), typhoid carrier responsible for multiple outbreaks of typhoid fever. Mary immigrated to the United States in 1883. In 1906, after six people became sick with typhoid in a household of 11 where Mary had worked as cook, the New York City Department of Health sanitary engineer George Soper investigated the outbreak and met with Mary linking her to all 22 cases of typhoid fever that had been recorded in New York City and the Long Island area. Mary was committed to an isolation center on North Brother Island, part of the Bronx, New York. In 1910, the health department released her on condition that she never again accept employment that involved the handling of food. When an epidemic broke out in 1914 at a sanatorium in Newfoundland, New Jersey, and at Sloane Maternity Hospital in Manhattan, New York , Soper returned Mary to North Brother Island, where she remained until her death in 1938. Fifty-one original cases of typhoid and three deaths were directly attributed to Mary (more were indirectly attributed), although she herself was immune to typhoid.

This brief footnote is adapted from the Encylcopedia Brittanica whose original article wast revised and updated by Kara Rogers, Senior Editor.

World Health Organization (WHO), Report on the origin and spread of Covid –19, 2021.